RADIOGRAPHIC ANALYSIS OF PULMONARY VASCULAR DISTRIBUTION: A REVIEW*

CARL E. RAVIN, M.D.

Department of Radiology
Duke University Medical Center
Durham, North Carolina

ALTERATIONS in the distribution of pulmonary vascularity, as recorded on standard chest radiographs, provide significant clues to underlying pathophysiologic abnormalities in the cardiopulmonary system. Although the radiograph is a static image, with a clear understanding of basic physiologic principles governing distribution of blood in the pulmonary vascular bed, much can be surmised about dynamic processes in the lungs at the time the film was exposed. Indeed, recognition of certain characteristic radiographic patterns of pulmonary vascular distribution may allow a relatively specific diagnosis.

THE NORMAL PULMONARY VASCULAR BED

The pulmonary vascular bed is a low resistance, low pressure system unique among vascular beds in the body in that it is the only one to receive the entire ventricular output with each systolic contraction. The cardiac output can vary from a normal of approximately 5 liters per minute[1] to as much as 40 liters per minute during strenuous exercise.[2] Under normal circumstances, the pulmonary vascular bed is able to accommodate these large increases in cardiac output with little change in pressure. The implication of this is that a significant reserve capacity is normally available in the pulmonary vascular bed that can be "recruited" to handle increased loads. Recruitment opens previously nonperfused vascular channels, distention of vessels already perfused, or both.[3,4] It is likely that both factors play some role in accommodating increased loads in the pulmonary circuit, although at lower pulmonary artery pressures
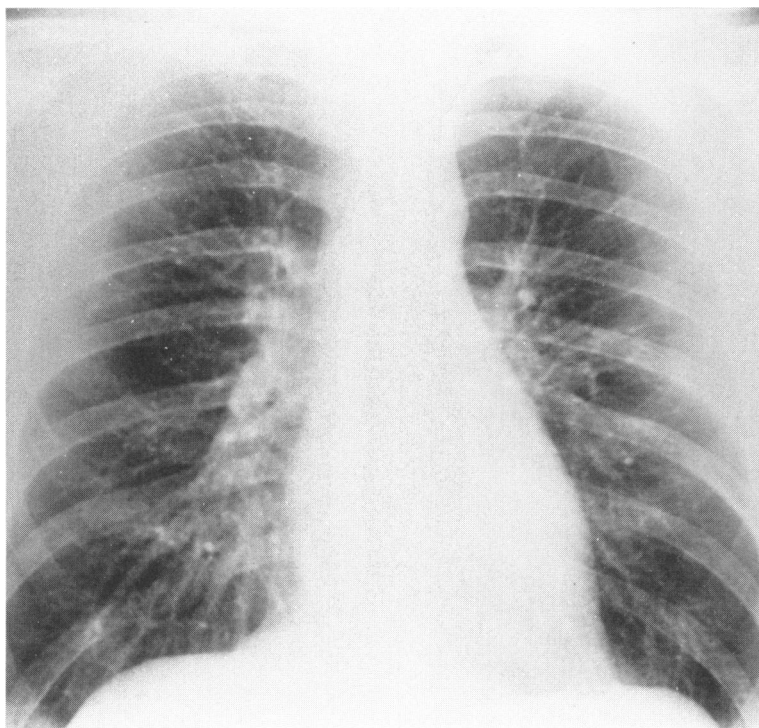

Fig. 1a. PA chest radiograph exposed with the patient standing erect. Note that the vessels supplying the upper lungs are approximately one third to one quarter the size of those in the lower lungs. This difference in size reflects the difference in pulmonary blood flow between upper and lower lung zones. Reproduced by permission from Ravin, C.E.: Observations on Pulmonary Vascular Distribution. In: *Pulmonary Diagnosis — Imaging and Other Techniques,* Putnam, C.E., editor. New York, Appleton-Century-Crofts, 1981.

opening of previously nonperfused vascular channels is probably the predominent mechanism.[4]

In man, when standing erect, there is normally an increasing gradient of perfusion per unit volume of lung tissue from apex to base. West and others have demonstrated that this gradient results from the effects of gravity superimposed on the low pressure, low resistance pulmonary vascular bed.[5-7] When these gravitational effects are removed by placing the patient supine, the flow per unit volume of lung becomes equal between apex and base. Such changes in distribution of pulmonary blood flow are routinely reflected in chest radiographs. In a standard chest film exposed at total lung capacity with the patient standing erect, there is normally a difference in vessel size and number from lung apex to base (Figure 1a). Vessels supplying the upper lung fields are smaller and fewer
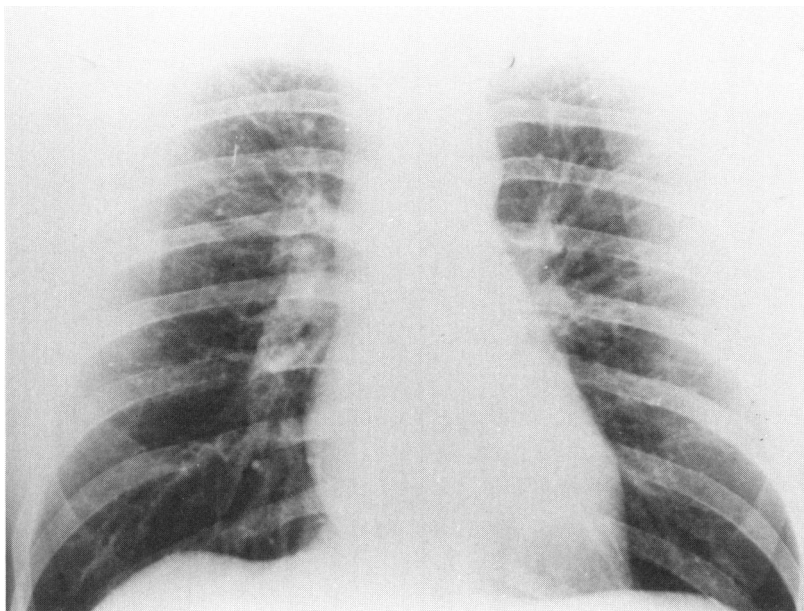
Fig. 1b. The same patient radiographed in a supine position now demonstrates more nearly equal size of vessels in the upper and lower lung. This change reflects more even pulmonary blood flow distribution between upper and lower lung zones. Reproduced by permission from Ravin, C.E.: Observations on Pulmonary Vascular Distribution. In: *Pulmonary Diagnosis — Imaging and Other Techniques,* Putnam, C.E., editor. New York, Appleton-Century-Crofts, 1981.

in number than those supplying lung bases. However, when the patient is supine, vessel sizes and numbers tend to equalize between upper and lower lung fields (Figures 1b, 1c). In fact, if the subject stands on his head, the normal gradient can be reversed and more flow (reflected as larger and more numerous vessels) in noted in the lung apex than at the lung base.

REDISTRIBUTION OF FLOW

The radiographic and physiologic observations noted above suggest that in man, when standing erect, a significant component of the available pulmonary vascular reserve exists in the upper lung zones. This reserve can be recruited to handle increased volume loads in the pulmonary vascular bed, and, therefore, recognition of an alteration in normal pulmonary vascular distribution, particularly of increased flow through the upper lung fields, can be a significant clue to an underlying pathophysiologic abnormality in the cardiopulmonary system. Increase in flow is recognized

in chest radiographs by an increase in the size and number of vessels supplying a given area of the lung.[8,9] An increased load requiring vascular recruitment can result from either an actual increase in the amount of blood contained in the pulmonary vascular bed (an increase in content) or from a decrease in size of the available pulmonary vascular bed (a decrease in capacity). Either situation results in a relative increase in the load that the pulmonary vascular bed must handle, and this triggers recruitment of any available pulmonary vascular reserve. Such recruitment is most easily recognized in chest radiographs as an increase in the size and/or number of vessels supplying the upper lung fields, i.e., redistribution. The earliest recognizable level of redistribution occurs when perfusion of upper and lower lung zones is equal.[10] As redistribution becomes more pronounced, the size and number of vessels in upper lung zones may actually exceed that seen in lower lung zones.

While recognition of redistribution of flow is subjective in most cases, based on the observer's experience in analyzing the size and number of vessels normally seen in the upper lung zones, several more objective criteria may help to substantiate the diagnostic impression. For example, in the perihilar regions, an artery is often seen "en foss" adjacent to its companion bronchus. Normally, the diameter of the artery is the same as that of the companion bronchus. When redistribution occurs, the artery will appear larger than the accompanying bronchus.

Dilatation of upper lobe veins occasionally may also be convincingly demonstrated, particularly in the lateral view. On the left lateral view, the upper lobe veins generally are anteroinferior to their corresponding arteries, and frequently the anterior vein of the right upper lobe is clearly visualized. Chang[11] pointed out that when the diameter of the right anterior vein is more than one and one half times that of the right anterior segmental artery, the upper lobe veins should be considered dilated and redistribution of flow present. While this sign may occasionally be helpful, we have found that it is frequently difficult reliably to distinguish arteries from veins. However, the lateral view frequently confirms redistribution suspected from the frontal projection. In such cases, vessels seen in the upper lungs are often much larger than at the lung base, and changes may be much more convincing on the lateral view.

The above considerations apply to situations in which the radiograph is exposed at full inspiration (total lung capacity) with the patient standing erect. As previously noted, placing the patient supine removes gravitational differences between lung apex and base, and results in equalization of

flow between these two regions. This is probably the most common cause of apparent redistribution of flow in daily clinical practice.

Failure to expose the film at full inspiration can also result in apparent redistribution of pulmonary blood flow. This is thought to occur because the caliber of arteries and veins running through the lung parenchyma is greatly affected by lung volume.[4,12,13] Expansion of the lung determines the extent to which the lung parenchyma pulls on the walls of the arteries and veins. When the lung volume is decreased, pull on the vessel wall is also decreased and vessel caliber is reduced. This has the effect of reducing the overall cross-sectional area of the pulmonary vascular bed, which in turn triggers recruitment of previously unused vascular reserve to handle the relatively increased load. As with other forms of recruitment, this is often most easily recognized as increased flow, relative to normal, through the upper lung fields. In addition, because many changes related to lung volume occur first at the lung base, selective diminution of size in vessels in this area causes some obligatory redistribution of flow to the upper lung fields. Therefore, films exposed at lung volumes significantly less than total lung capacity tend to show pulmonary vascular congestion and apparent redistribution of blood flow.

Once redistribution of flow is recognized and supine positioning or suboptimal inspiration excluded, a reasonable differential diagnosis can be generated by analysis of associated radiographic features. The major categories in the differential include diseases producing pulmonary venous hypertension, pulmonary arterial hypertension, increased right ventricular cardiac output, and pulmonary parenchymal destruction. Each of these categories and its associated radiographic features will be considered below.

PULMONARY VENOUS HYPERTENSON

The classic cause of redistribution (i.e., increased vessel size and number in the upper lung fields) in routine chest radiographs is pulmonary venous hypertension. The most common causes of venous hypertension are chronic left ventricular failure and mitral valve stenosis.[14-16] In both situations redistribution of flow may be quite marked, the size and number of vessels in the upper lung fields actually exceeding that observed at the lung bases. When pronounced, such findings are very striking, and reflect not only recruitment of available pulmonary vascular reserve in the upper lung fields but associated constriction and diminution of the pulmonary
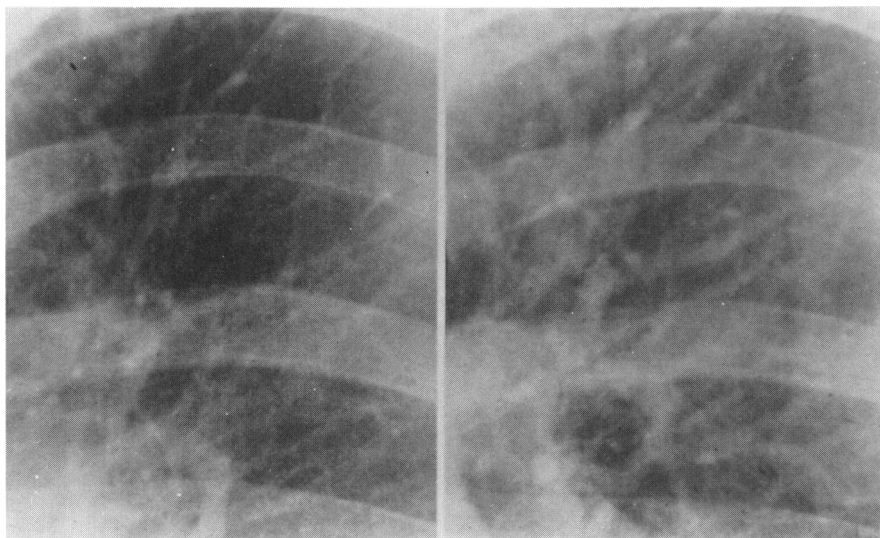
Fig. 1c. A composite view of the left upper lung with the patient standing erect (left hand side) and the patient lying supine (right hand side) to show the change in size and number of vessels accompanying change in position. Reproduced by permission from Ravin, C.E.: Observations on Pulmonary Vascular Distribution. In: *Pulmonary Diagnosis — Imaging and Other Techniques*, Putnam, C.E., editor. New York, Appleton-Century-Crofts, 1981.

vascular bed at the lung bases. Generally, when such diminution occurs, the underlying pathologic process has been present for a long time, as opposed to acute hemodynamic change. The mechanism by which the basilar vascular bed becomes reduced in size has not been fully explained and remains controversial.[8,17,18] Nonetheless, the radiographic appearance is relatively characteristic, with large vessels in the upper lung zones and a relative decrease in size and number of vessels in the lower lung fields (Figures 2a, 2b).

While the most striking changes of redistribution are noted in chronic settings, less severe changes may be noted with milder elevations in pulmonary venous pressure. This occurs most often in left ventricular failure. As venous pressure rises, redistribution of flow increases and vessels in the upper lung fields become equal in size to those in the lower lung fields.[19,20] In general, such changes are observable when the mean pulmonary wedge pressure exceeds 15 mm. Hg. When pulmonary vascular pressure exceeds plasma oncotic pressure (normally approximately 25 mm. Hg) fluid begins to leak from the vessels into the adjacent interstitial tissues and a septal line may become evident. Continued leakage of fluid
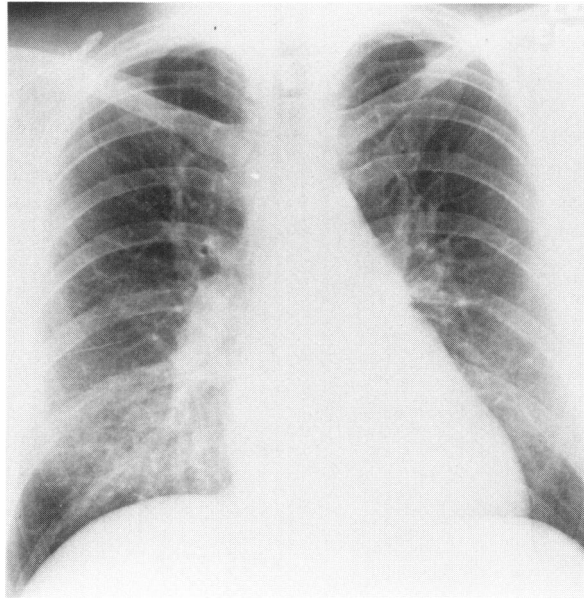
Fig. 2a. Patient with mitral stenosis. Note that the vessels in the upper lobe are as large and numerous as those at the lung base. This reflects redistribution of flow to the upper lung fields.

into the interstitium, if not compensated for by lymphatic clearance, eventually leads to alveolar edema. Although it is theoretically possible to predict capillary wedge pressure from careful analysis of the radiograph, prospective studies have shown the accuracy of such predictions to be variable.[21,22] Nonetheless, recognition of equalization of flow between upper and lower lung zones is a good sign of underlying pathophysiologic abnormality and, when venous hypertension is the cause, reflects pulmonary capillary wedge pressure in excess of 15 mm. Hg.

PULMONARY ARTERIAL HYPERTENSION

While redistribution of flow secondary to pulmonary venous hypertension has long been recognized, convincing demonstration of redistribution secondary to pure pulmonary arterial hypertension is a more recent observation.[23] Proof of such redistribution centered around observations in patients in whom concomitant elevation of pulmonary venous pressure had been excluded. Also excluded were patients in whom the pulmonary vascular bed at the lung base might have been selectively destroyed by underlying disease because such destruction would result in an obligatory
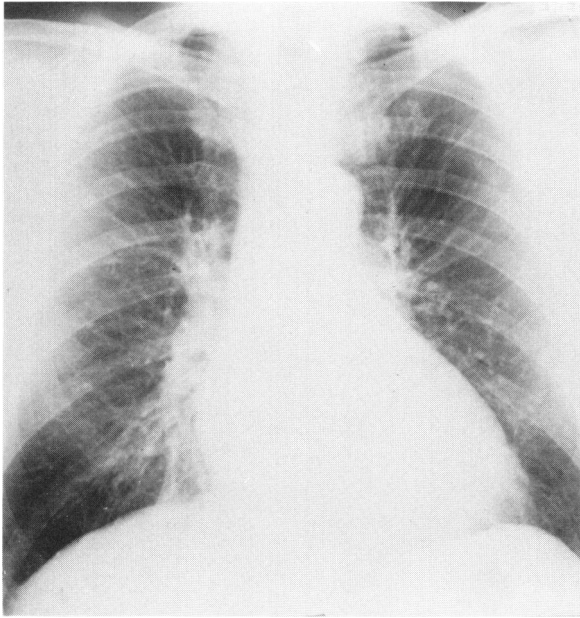
Fig. 2b. Patient with congestive heart failure. Note that the size and number of vessels in the upper lung zones are increased relative to normal. They are virtually equivalent to or slightly exceed, in both size and number, the vessels seen at the lung base. This pattern of pulmonary blood flow is associated with chronic elevation of pulmonary venous pressures.

redistribution of pulmonary blood flow to the upper lung zones. Two groups of patients have been identified in whom pulmonary arterial hypertension exists in a pure form: those with central alveolar hypoventilation (Ondine's curse)[24,27] and those with primary pulmonary hypertension.[28,29] In both these processes the vascular bed is affected diffusely, and specifically there is no tendency for selective involvement of the vascular bed at the lung bases. In addition, pulmonary wedge pressure in both groups of patients is normal, excluding concomitant venous hypertension.

Patients with central alveolar hypoventilation suffer dysfunction of the automatic control of respiration which leads to generalized hypoventilation of the alveolar spaces. The resulting low intra-alveolar oxygen tension causes vasoconstriction diffusely throughout the lungs, produces a significant reduction in cross-sectional area of the pulmonary arterial bed, and thus causes pulmonary arterial hypertension. The pulmonary parenchyma, exclusive of the vascular system, is otherwise completely normal.
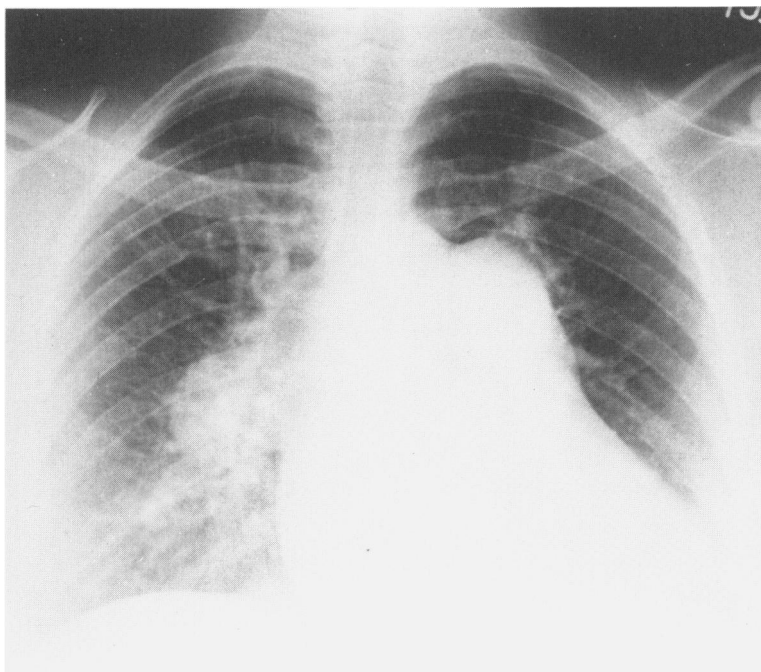
Fig. 3. Patient with primary pulmonary hypertension. Note the striking prominence of the main and central pulmonary arteries as well as the equal size of vessels supplying the upper and lower lungs. There is apparent equalization of vessel size and number compatable with the equalization of pulmonary blood flow between upper and lower lung zones seen in this patient group. There is also a tendency for the vessels in the periphery of the lung to appear smaller than those centrally suggesting a "centralized" pattern of pulmonary blood flow. Reproduced by permission from Ravin, C.E.: Observations on Pulmonary Vascular Distribution. In: *Pulmonary Diagnosis — Imaging and Other Techniques,* Putnam, C.E., editor. New York, Appleton-Century-Crofts, 1981.

Patients afflicted with primary pulmonary arterial hypertension also suffer a generalized reduction in cross-sectional area of the pulmonary arterial bed. The cause of the disease has not been clearly established, although it is recognized that there is a significant reduction in the lumen size of the small pulmonary arteries and arterioles caused by muscular medial hypertrophy and intimal proliferation.

Radiographs obtained from both these patient groups demonstrate redistribution of flow as evidenced by an equalization of vessel size between upper and lower lung fields (Figure 3).[23]

Patients with pulmonary arterial hypertension differ from those with pulmonary venous hypertension in that, in addition to redistribution, they

also demonstrate enlargement of their central pulmonary arteries. The changes are noted in both the main and central pulmonary vessels and there is an obvious discrepancy between the enlarged central pulmonary vessels and the peripheral vessels, which appear narrowed. Chen et al.[32] have referred to this particular radiographic appearance as a "centralized" pulmonary blood flow pattern. We have found that it is relatively specific for pulmonary arterial hypertension whatever the underlying etiology.

Doppman and Lavender[33,34] have suggested that diseases that primarily enlarge the pulmonary arteries (e.g., pulmonary arterial hypertension) can be distinguished from those which primarily enlarge the pulmonary veins (e.g., pulmonary venous hypertension) by careful analysis of the right hilum. The normal right hilum can be divided into upper and lower halves. The upper half is composed mainly of the descending right upper lobe vein and the lower half is primarily formed by the main trunk of the descending pulmonary artery. An angle is formed where these vessels overlap, which divides the hilum into its two components. Enlargement of the pulmonary veins, as seen in pulmonary venous hypertension, will theoretically obliterate the normal hilar angle and provide a useful sign of pulmonary venous hypertension. In our experience, this radiographic sign has occasionally proved helpful, but in some cases pure pulmonary arterial hypertension mimics the appearance of venous hypertension.

INCREASED PULMONARY BLOOD VOLUME

The best known cause of increased pulmonary blood volume is shunting of blood from the left side of the circulation to the right (left-to-right shunt). Such shunts increase pulmonary blood volume relative to normal. This, in turn, necessitates recruitment of available pulmonary vascular reserve to accommodate the increased load. As with other forms of recruitment, one observes increased flow through the upper lung fields as evidenced by an increase in number and size of vessels seen in the upper lung zones. The pattern seen with left-to-right shunts differs from the two patterns discussed previously in that, prior to the development of Eisenmenger's physiology, all vessels appear enlarged (Figure 4a).[35] That is, while size and number of vessels in the upper lung fields are obviously increased, a similar increase is noted throughout the lung fields including the lung bases. In addition, vessels appear to extend further toward the periphery of the lung than is normally seen. Although left-to-right shunts are the classic cause of increased blood volume, a similar, although
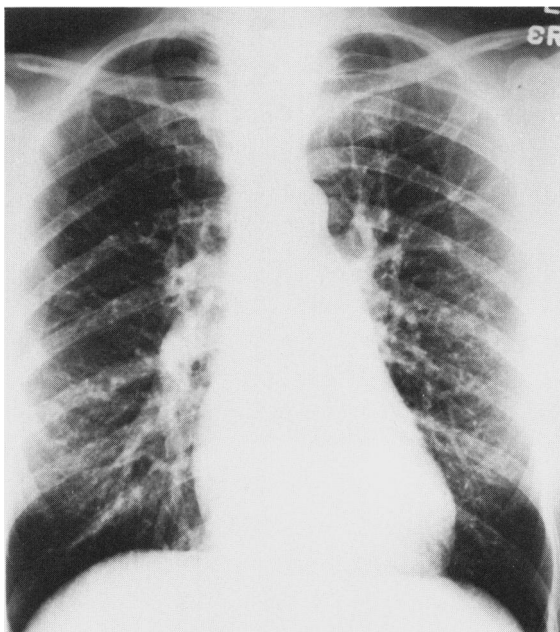
Fig. 4a. Patient with an atrial septal defect. Note that while the vessels in the upper lung zones are larger and more numerous than normal, all vessels throughout the lungs are increased in size. This radiographic pattern of a generalized increase in pulmonary vessel size and number is associated with increased pulmonary blood flow.

generally less pronounced, effect can be seen with any cause of increased right ventricular output. Such situations occur following heavy exercise[36] or with high fever, severe anemias, (Figure 4b) thyrotoxocosis,[1] pregnancy, or in some patients with heart block[37] in whom the slow heart rate results in large stroke volumes.

Eisenmenger's physiology is a reaction to continued large volumes of blood coursing through the lungs. The peripheral arterioles begin to constrict and the gradual onset of pulmonary arterial hypertension results. Concomitant with this change, the radiographic pattern begins to shift from that seen with a pure left-to-right shunt toward that seen with pulmonary arterial hypertension. That is, the central pulmonary arteries become large and the peripheral vessels appear disproportionately small. Such changes may be extremely striking in the latter stages of the disease process. In the end stage of the disease, it may be impossible to distinguish those changes seen with left-to-right shunts and subsequent development of Eisenmenger's physiology from those seen with pure pulmonary arterial hypertension.

Fig. 4b. Patient with sickle cell anemia. Because of the severe anemia, the patient has a hyperdynamic cardiac circulation and the radiographic pattern mimics that seen with a left-to-right shunt. In this case, the volume of fluids circulated through the lungs is increased over normal giving an appearance similar to a shunt.

PULMONARY PARENCHYMAL DESTRUCTION

If the pulmonary vascular bed is reduced in overall size and right ventricular output remains normal, a relative increase in blood volume results. Recruitment of available vascular reserve is necessary to handle the relatively increased load. The most common cause of such changes is emphysema (Figure 5). In these cases, parenchymal destruction is usually generalized and, when severe enough, can cause pulmonary arterial hypertension. In most cases, redistribution of flow reflects concomitant pulmonary arterial or venous hypertension or both, although it is theoretically possible for obligatory redistribution to result from destruction of the parenchymal bed alone prior to the development of a hemodynamic abnormality. On occasion, selective destruction at the lung bases by the disease process can produce very striking redistribution of flow on an obligatory basis.

Fig. 5. Patient with emphysema. There is considerable destruction of the pulmonary parenchyma and associated vascular bed by the disease process. This has resulted in some obligatory redistribution of flow from the lung bases to the upper lung zones. The diversion of pulmonary blood flow is reflected in increased size and number of vessels in the upper lung zones compared to normal. In addition, there is mild prominence of the central pulmonary arteries suggesting mild pulmonary arterial hypertension.

The radiographic clue that redistribution occurs secondary to destruction of the pulmonary vascular bed by emphysema is the somewhat patchy nature of the radiographic changes. The normal dichotomous vascular branching pattern is often distorted, and focal areas may exhibit fewer vessels than are seen in other comparable areas. In addition, the more classic signs of obstructive lung disease, including flattening of the hemidiaphragms, increase in retrosternal air space, and hyperaeration of the lung fields are generally present.

The size of the pulmonary vascular bed also may be significantly reduced following direct occlusion of pulmonary arteries by pulmonary emboli. A number of physiologic studies in animal models have demonstrated that approximately 70% or more of the vascular bed must be

occluded before a rise in pulmonary artery pressure is observed.[38] However, if of sufficient size or number, pulmonary emboli may reduce the overall cross-sectional area of the pulmonary arterial bed so significantly that recruitment of normally unused portions of the pulmonary arterial bed becomes obvious. The appearance of recruitment is enhanced by the fact that emboli tend to go to the areas of greatest blood flow and thus are more commonly directed to the lung bases. Occlusion of the pulmonary vascular bed at the lung base will result in some obligatory shift of flow to the upper lung fields. While it may be difficult to identify with certainty the cause of such redistribution, in certain cases associated vascular spasm and resultant oligemia (Westermark's sign) suggest the correct diagnosis.

SUMMARY

Decrease in the size of the pulmonary vascular bed or increase in pulmonary blood volume necessitates recruitment of available pulmonary vascular reserve to handle the relative increased load. Such recruitment is recognized as "redistribution" of pulmonary blood flow as evidenced by an increase in size and number of vessels in the upper lung zones. Observation of redistribution is a definite sign of underlying cardiopulmonary pathophysiologic abnormality. Clues to the specific underlying etiology can be obtained from recognition of associated findings. In the setting of chronic venous hypertension, increased flow in the upper lung fields is associated with a diminution in vessel size at the lung bases. When redistribution occurs secondary to pulmonary arterial hypertension, there is concomitant increase in size of the central pulmonary vessels and an equalization of flow between upper and lower lung fields. In addition, there is a relative diminution in the size of the peripheral pulmonary vessels. When there is a true increase in the amount of blood in an otherwise normal pulmonary vascular bed (as seen with left-to-right shunts or other causes of increased right ventricular output), redistribution is associated with increased flow throughout the lung fields as reflected by larger vessels at the lung bases as well as in the upper lung zones and more peripheral extension of vessels than is normally seen. Finally, destruction of the pulmonary parenchymal bed by diseases such as emphysema is generally accompanied by the radiographic hallmarks of this disease, and may be suggested by the focal nature of vascular destruction seen in the lung fields.

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